tracheo-bronchial nodes, but would tend to retain them in the lungs of the rabbit. Krause states that while the bloodvessels are potent factors in the spread of infection, they act merely as conveyers of bacilli. The real site of the pathologic development of tubercle would appear to be in the lymphatic system, in accordance with the view of Calmette. From mucous membrane the bacillus makes its way into the lymphatic system; and from plugged capillary it does likewise. Where it will focalize is determined largely by lymphatic anatomy (and, therefore, lymphatic physiology); and variations in lymphatic anatomy will determine differences in focalization. Krause has shown that fundamental anatomical differences are actualities which give point to Tendeloo's views of tuberculous focalization, based on physiological inquiries; though he is not inclined to follow Tendeloo all the way, and especially not in regard to non-continuous infection against the lymph stream. With this experimental evidence, he asks a few questions which continually recur to him but which thus far he has not had time or opportunity to investigate in the thorough way which their solution would require. Does human tuberculous infection normally tend to the lung root in early life because then lymphatic drainage is more active and intrapulmonary channels more open and unobstructed than later? In other words, is the child's lung more comparable to that of the guinea-pig, which may be looked upon as constituting one type? With increasing age, do human lungs undergo internal changes which bring them nearer to the type which is normal for rabbits? Does there normally occur an increase and development of intrapulmonary lymphoid tissue over a few years, or through many years? Do formerly open lymphatic paths thus become more or less obstructed, so that bacillary retention within the lung is favored? How much do the inhalation of foreign bodies and the repeated occurrence of respiratory infections alter the puerile lymphatic structure which was once normal? Gardner and Willis have shown that in guinea-pigs the intrapulmonary lymphoid tissues proliferates markedly in response to the irritation of dusts, and that these overdeveloped masses then remain as permanently enlarged structures in the lymphatic paths. Miller has described and figured the same occurrence in man. Dust which is not cast out by the ciliated epithelium comes to be laid down in human lungs along the lymphatics. With increasing age fibroses mark our lungs in varying degree. How much have all these processes to do with bringing about anatomical changes which will tend to arrest bacilli in their lymphatic passage to the nodes and to fix them in the lung itself? Is it not remarkable that even when the adult human lung has massive tuberculosis, the root nodes participate relatively little in the process? Here we have a companion piece to what is normal for the rabbit. What has blocked the relatively easy passage to root which characterizes childhood infection? Krause thinks it is not wholly improbable that the several parts of the lung may differ in details of finer lymphatic structure—in details not fundamental perhaps, yet decisive as regards fixation of bacilli—throughout life or perhaps only at certain periods. May the apical occurrence of localized adult tuberculosis, which is due to relatively slight infection, dependent on such factors? Do we lose our general tendency to lymphatic tuberculosis as we grow older, because with increasing age our lymphatic flow becomes less active and our lymph nodes more impermeable? A rising incidence of renal and epididymal tuberculosis suggests that age brings no diminution of blood-borne metastases; but wherever the lymphatic apparatus may be conceived as entering into the spread of infection, there is in general much less tuberculosis as childhood passes into adolescence, adolescence to full maturity and completed growth to old age. Krause leaves all these as questions, as phases of a much larger problem, which, he submits, is worthy of attack from the point of departure which he has tried to establish. This is that the determinants of tuberculous infection are almost innumerable; yet among them there exists a very fundamental one which will always bulk large in the shaping of the final result and is comprised in the fact that a varying and variable anatomical structure establishes the loci of infection and to a great extent the further development and progression of infection from these.

Relation of Faulty Cultures to Diphtheria Mortality.—O'KEEFE (*Boston Med. and Surg. Jour.*, 1922, **186**, 603) states that immediately following the common use of diphtheria antitoxin, and after the more general use of bacteriologic diagnosis, the mortality curve of diphtheria took a marked downward trend. As the value of antitoxin became more widely recognized by the profession and public, this curve went lower still. Carey states that in the first decade of the last twenty years the death rate of this disease fell from 43 per 100,000 to 15 and a fraction. He goes on to state that there has, however, been a fairly constant mortality rate during the latter ten years of this twenty-year period. With the advent of toxin-antitoxin administration it is not unreasonable to expect, in the near future, still further lowering of the diphtheria death rate. O'Keefe concludes from his observations that one of the factors in delay of diagnosis of diphtheria is the failure to recognize that unless cultures are properly taken laboratory diagnosis will not be reliable. No amount of care or skill on the part of the bacteriologist can compensate for faulty technic on the part of the practitioner in securing a fair sample of the organisms existing in the membrane or exudate concerned. Twenty-four to forty-eight hours' delay in the administration of antitoxin frequently results, owing to reliance placed upon faultily taken cultures.

Tularemia Francis 1921, Six Cases of Tularemia Occurring in Laboratory Workers.—LAKE and FRANCIS (*Public Health Reports*, 1922, **37**, 392) report on 6 cases of laboratory infection occurring in laboratory workers. Their paper is summarized as follows: All of the persons (6) who have been intimately engaged in the past two years in the laboratory in handling or dissecting rodents infected with the Utah strains of tularense have suffered an attack of tularemia. The diagnosis in each of the 6 cases rests upon the occurrence of a febrile period lasting about three weeks, positive serum reactions for agglutination and complement-fixation to antigens composed of Bacterium tularense and the absence of febrile attacks in 100 other persons in the laboratory coming in casual contact with infected rodents. Consideration must be given to the skin of the hands as a possible portal of entry of the infection in laboratory workers even in the absence of local lesion or lympha-